

ON A CASE OF SUBCRANIAL HÆMORRHAGE  
TREATED BY SECONDARY TREPHINING.

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ON Thursday, June 16, '87, a laborer, named Patrick Rourke æt. 50, of robust habit and sanguine temperament, was received into the Richmond Hospital. He was reported to have been drinking heavily up to the Sunday previous to his admission, and on that day to have fallen off a cart and sustained such injury that he became insensible. No accurate history of his condition between the Sunday and Thursday could be obtained, or no hint as to whether his fall was the result of previous loss of consciousness, or his loss of consciousness incident on his fall. The possibility of violence having been used was present, and was supported by the apparent disinclination of those who had been in contact with him to give information. The most we could gather of his condition during the interval between his injury and his admission to hospital was that he had been more or less stupid, and apparently suffering from the effects of his previous debauch.

This absence of any reliable history complicated the difficulties of the case, and was one of the reasons why operation was delayed so long.

At the time he came under observation, on the fourth day after the accident, his condition was as follows:—He was in a state of stupor, from which he could easily be aroused, when he made slow but sufficiently intelligent answers. His pupils were of average size, symmetrical, and fairly responsive. He had very slight partial paralysis of his left facial nerve, with

the usual protrusion of the tongue to that side; there was partial motor paralysis of the left arm, and a barely detectible motor insufficiency of the left lower extremity. He swallowed well, and his speech, though thick, was quite intelligible. There was no sensory paralysis.

On his head being shaved and examined, a bruise was found on the right side, of the cranium, 25 mm. from the mesial line, on that part of the scalp which would correspond to the upper part of the fissure of Rolando. It was about 20 mm. in diameter and indurated round the edge, so that it was not possible to say whether a depressed fracture existed, or merely a ring of hardened blood-clot. In consultation it was determined to delay operative treatment pending the persistence or farther development of symptoms, as there was not sufficient evidence as to whether the cerebral pressure present might not be apoplectic.

On the next day there was but little change, except that stupor was more marked; but on that following, the sixth after the fall, there was considerable change for the worse. He could with great difficulty be aroused. The breathing was stertorous. The right pupil was slightly larger than the left. The paralysis of the left arm was nearly complete, while that of the lower extremity, though incomplete, was more evident than at first.

On the next day, June 19, the seventh of the case, his condition was rather improved; but on the night of the eighth day he got rapidly worse, and when I visited him on the morning of the ninth day after the accident, his state was as follows:—The face and extremities of the left side and the bladder were completely paralyzed. The pupils were symmetrical, and, without being actually dilated, were rather over medium size. He was in a state of profound coma, and the automatic act of swallowing was performed in the case of small quantities of fluid with marked slowness and imperfection. The breathing was slow, 12 to 14 in the minute, and was stertorous and diaphragmatic. The temperature, which had previously been at or about the normal point, except on the sixth day, when it went up to 100°, had fallen to 98°; and the pulse, which had for some days been gradually increasing in

frequency, had risen to 110. The man was evidently failing rapidly, and would die soon if unrelieved.

Looking at the entire view of the case, in spite of its obscure history, I came to the conclusion that the symptoms were probably due to hæmorrhage between the bones and dura mater, dependent on a laceration, with or without fracture, of the middle meningeal artery. Although there had been, so far as we could ascertain, no complete recovery between the injury and the advent of the later symptoms of pressure, and therefore an absence of that interval which is so characteristic of subcranial hæmorrhage, yet, on the other hand, there had not been that even level of ills, or that uniformly downward path of symptoms, indicative of laceration or apoplectic pressure on the brain.

The reading of the case, as expressed at the time, and fully borne out by its subsequent history, was this:—

1. Hæmorrhage over the right motor area, between the bone and dura mater, probably due to laceration of the middle meningeal artery or one of its branches, most likely associated with fracture, and producing the partial left paralysis which at first existed.

2. Subsequent increase of the hemiplegia, either due to renewed hæmorrhage, or to that sudden yielding of brain function which is repeatedly seen both in hæmorrhages or serous effusions which have existed for some time, even though no additional mechanical pressure is called into play.

Let us examine the reasons for these conclusions.

The existence of the scalp injury over the upper and back part of the motor area was of less value in indicating the seat of pressure than the opposite paralyses, which pointed clearly to the engagement of the greater portion of the right motor area, including the extensive surface occupied by the cortical centres for the various parts of the upper extremity, the face and tongue, and lower extremity, the interference with them being in the sequence in which I have written them. Remember, in this context, the statement of M.M. Charcot and Pitres (*Revue de Médecine*, October, 1883), more lately repeated and endorsed by Ferrier, that "there does not exist a single accurate observation of a destructive lesion outside the motor

area having produced permanent paralysis; nor does there exist a single accurate observation of a destructive lesion of any extent of the ascending convolutions which has not given rise to permanent paralysis of the opposite side of the body."

The dilatation of the pupil on the same side of the cerebral lesion is the condition most usually found, and the disappearance of the mydriasis when the other pressure symptoms became aggravated is but an example of that compensation derived from the centres of the opposite hemisphere, as shown by the law of Broadbent, which teaches that "one-sided movements are represented in both hemispheres, and can be excited from either, in proportion as they are habitually associated on the two sides" (Gowers). The eyes have an intimate association of action which would cause this rule to apply to them more strongly than to other parts.

As to my assumption that the pressure was cortical, and not apoplectic, it was founded on my belief in the absence of any sensory paralysis, and the teaching on that point, so well expressed by Ferrier, who says that "Strictly cortical lesions of the motor area do not cause anæsthesia in any form, and it may be laid down as a rule, to which there are no exceptions, that if anæsthesia is found along with motor paralysis, the lesion is not limited to the motor zone, but implicates also, organically or functionally the sensory tracts of the internal capsule or the centres to which they are distributed." The cortical nature of the pressure was farther supported by the existence in the early stage of the case of a pronounced brachial monoplegia, as it is well established that monoplegia is a condition due to interference with the cortex and not usually found in more deeply situated lesions.

The impeded functions of the respiratory centre and pneumogastric nerve, as evidenced by the increasing rapidity of pulse, the diminishing frequency of respiration, and the extreme difficulty of swallowing, were believed to be due to pressure on the medulla from above, rather than to any direct blood-pressure on the origins of the nerve, as, if blood had found its way so far as the under surface of the medulla, it would most likely have penetrated to the spinal canal and so relieved its pressure effect on the pneumogastric origins; be-

sides, the late and gradual advent of the pneumogastric symptoms was more likely due to cumulative pressure from above than to so late a secondary hæmorrhage as this should be, were it the direct cause of pressure, remembering that the medullary impediments did not exhibit themselves until the ninth day after the injury. This was rather an important point in determining operation, as a surgeon would naturally be slow to use the trephine if he believed basic extravasation to have taken place.

Considering the whole story of the case, and in view of the inevitable death of the patient if not relieved by treatment, it was determined to trephine him. This was done June 21, the day on which he exhibited the pronounced conditions just described, and the ninth after his accident.

The patient was completely comatose, and no anæsthetic was used. Taking the injury over the fissure of Rolando as the point indicated for operation, because, although not in the centre of the engaged portion of the motor area, a possible fracture existed there, two incisions were made, one downward and outward behind it, the other running horizontally forward, so as to include a rectangular flap. The scalp and pericranium were raised together, and the bone found to be free from any appearance of injury. A trephine, with a diameter of 26 mm., was applied, and on the disc of bone being removed, the antero-inferior edge of the opening disclosed the edge of a well-formed blood-clot, evidently reaching downward and forward over the parietal area. The incisions were extended, so that each measured 60 mm., and a second trephine opening was made immediately below and in front of the first. The projecting tongues of bone between the two openings, which ran into each other, were then cut off with a bone-forceps, and an oval opening measuring 52 mm., rendered available for removing the clot. This was effected by a director, the finger, and a current of sublimate solution, and a cavity between the bone and dura mater emptied, which was circular in outline, covered all that portion of the motor area which lay anterior and inferior to the trephine hole, and was at least 90 mm. in diameter; at its centre the clot was so thick that the dura mater was distant from the cranium about 40

mm. When this stage of the operation was completed, the dura mater began to rise towards the cranial wall, so that when the wound was sutured it was within 6 or 7 mm. of it. Signs of returning brain power at once showed themselves, and before the patient was taken off the operating table he moved his left arm and leg with tolerable freedom, asked for a drink of water, which he readily swallowed, and gave vent to some rather bad language.

Some little oozing of fresh blood took place, but gave no trouble. Strict antiseptic measures were observed; the solutions were of corrosive sublimate, 1 to 3,000, and the spray carbolic acid. The wound was sutured, except at its angle, where a drainage tube was inserted, and dressed with sublimated gauze and iodoform cotton. The dressing was changed daily, the drainage removed on the third day after operation, and the sutures on the fourth. An uninterrupted good recovery was made, pus appeared freely on the sixth day, but without any odor, and continued to be discharged for two weeks longer, when the discharge ceased, and the wound closed. The temperature, which was 98° on the morning of operation, rose on four occasions during the ensuing ten days to 100°, but, for the rest of the time the man was under treatment, was practically normal. The evening of the day of operation the paralysis and brain symptoms had all but disappeared, and he could pass water voluntarily. A day later he was in a perfectly normal condition as regards any brain symptoms. He was kept under observation until September 20, when he left hospital. He again presented himself on November 10, when he had a sinus discharging at the angle of the original wound. I received him into hospital, and the next day removed a small fragment of bone from the trephine hole. It had evidently been separated from the inner table, and showed clearly from its form that a fracture of the vitreous table had existed. The sinus has since continued to discharge a gradually diminishing amount of pus, but I cannot find denuded bone, and his excellent general health is in no way impaired.

If another case, resembling this in the extensive separation of the dura mater from the bone, offers itself for treatment, I will be disposed to make one addition to the methods here

employed. That is, if a drain should be necessary, I will consider the propriety of making it as efficient as possible by forming a small trephine opening at the nearest accessible point to the lower edge of the line of separation between the cranium and dura mater. The more perfect drainage thus afforded would, I believe, promote the healing of the cavity, and lessen the risks of suppuration, by offering a free and depending exit to pus or any other fluid present.

The case had some features of great interest. Not the least among these is the lesson it teaches as to possibility of making an accurate diagnosis, even in an instance so obscured by want of definite history as this was. There is nothing more clear than the justice with which Mr. Hutchinson, in his "Lectures at the London Hospital," points to the enormous difficulty which often exists in distinguishing compression, from laceration of the brain due to one cause or another. I escaped this dilemma and was able to arrive at a conclusion justifying a useful operation by two circumstances—first, that I could, independent of any knowledge of a fracture enabling me to localise the hæmorrhage, put my finger over the motor area and say with sufficient accuracy, "there is pressure here over the cortical centres for the upper extremity; it extended downward and forward to those for the face; it afterward reached upward to those for the lower extremity." The sequence is anatomically perfect. First a brachial monoplegia: then, as the blood or pressure effect extends, a faciolingual; and finally, a crural paralysis. Again, I was justified, using the knowledge of the fact that cortical lesions of the motor area do not cause anæsthesia in any form, and that no loss of sensation was present here at a time when the condition of the man permitted that fact to be tested, in assuming that the pressure was probably between the dura mater and bone; a decision to which I was farther helped by the monoplegic condition which at first was present. To what, I ask, do I owe the knowledge on which this judgment was founded? Largely to the humane and benevolent investigations of those biologists whom weak, credulous, or mistaken people are actively pelting with the verbal filth of prejudice and ignorance—people who would prefer that this man, formed in

the image of his Maker, should die, rather than their feeble sentiment be offended by a painless experiment on an ape.

Another point of interest is the very large size of the clot which was present, and the late development of the grave symptoms which were producing death. The clot was so uniform in color and consistence that I am persuaded it was all the result of primary or intermediate hæmorrhage, and that the failure of brain power on the ninth day was due, not to added pressure, but to the increased intravascular tension due to the vascular spasm produced by irritation of the sensory nerves or their centres, which, as Duret has shown, can destroy life before the amount of extravasation is reached which is capable by itself of producing a fatal disturbance of cerebral function.

As bearing on localisation of function, this case has an interest beyond the corroboration it affords to what is held concerning the seat of the cortical centres for the various parts of the upper extremity, and the situation of that facio-lingual area. It helps to prove what is generally believed as probable, that the centres for the leg do not extend behind the fissure of Rolando into the superior parietal lobule at the point indicated by Ferrier as No. 1 area. I have made the most careful measurements of this man's skull, both by the method of Reid and Thane, and I am satisfied that the centre of the upper trephine hole covered exactly the superior part of the fissure of Rolando 25 mm. from the median line. If it be remembered that the clot was found to extend downward and forward from the lower margin of this opening, it will be at once seen that it did not at its postero-superior limit approach within 10 mm. of the anterior edge of the fissure, and that the superior parietal lobule could not have been directly engaged. If it be urged in answer to this that the pressure producing the effect might be remote, it may be replied that in this case the remote pressure effect, that on the medulla, which undoubtedly did exist, seemed to expend itself downward in the direction of gravity, as might naturally be expected.

Independent of these points, the case, as one in which no defined bone lesion served to localize the hæmorrhage, belongs to a class sufficiently rare to deserve notice. If I required any



other apology for presenting it at such length, I would have it in the words of so eminent a surgeon as Mr. Hutchinson: who says, speaking of instances of effusion of blood between the bone and dura mater—"These are especially important, because generally supposed to be capable of relief by treatment. Yet it is a remarkable fact that the modern annals of surgery do not, so far as I am aware, contain any cases in which life has been saved by trephining for this state of things" ("Clin. Lect. Lond. Hosp.," 1867-68). The present is but one of two instances which have come under my own notice. In the *Med. Press and Circular* of Dec. 17, 1879, Mr. Thomson communicated the case of a child whom he had trephined in the Richmond Hospital during the early part of that year for primary subcranial hæmorrhage over the right parietal region, and who made an excellent recovery. There are others recorded, for I find in Ashhurst 40 cases collected, several of which have occurred within the last 20 years. Of these, 24 recovered and 16 died; 22 of the 40 were primary operations, and of these 8 proved fatal; 19 were secondary operations, and of these 8 recovered.

The lesson of this case has impressed itself deeply on my mind; for, in other practice as well as in my own, I have seen instances in which life might, as I now believe, have been saved by operation such as this. In one case in particular, where a trephine hole was made by one of my colleagues, it was proved afterward that the opening barely escaped the discovery of a clot, which a greater knowledge of localisation than we then possessed, and a second perforation, would have enabled us to discover and remove, with the probable effect of saving life.

On a careful study of the light which modern investigation has thrown on the localisation of intracranial pressure, and the security which modern surgical methods have given to the operation of opening the cranium, and bearing my clinical observation of deaths from doubtful intracranial accidents in mind, I have come to this conclusion for future guidance—that *if I am in doubt I will operate*; that whether I believe the blood to be subcranial or subdural, I will not, so long as evidence of such constitutional disease or local condition as

points to apoplexy is absent, allow a patient to die without giving him the chance of escape from a fate which appears inevitable if the surgeon holds back. Why should we hesitate to open the dura mater even if the bleeding be arachnoid? Are not the numerous cases which exist, of recovery when the brain has been exposed and extensively lacerated, sufficient answer to the policy of hesitation?

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## SOME POINTS ON INCARCERATED HERNIA.

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FOR many years the subject of incarcerated hernia has engaged my attention, not only because of its difficulties, but also because of its intrinsic interest: That it is a difficult subject, no one who has any knowledge will deny; and that it is of interest, any one will admit who considers the important field it offers for practical study. And it has seemed to me that it would not be out of place to occupy a little time with the consideration and the discussion of some of the difficulties of this form of hernia. And then I may be permitted to bring forward a few cases in illustration of my views and opinions, in regard to some of the most difficult questions of surgical practice.

In the first place by way of definition, let me say that an incarcerated hernia involves two essential conditions: 1. It must be irreducible: 2. It must be obstructed. These two conditions are essential and necessary in a case of incarcerated hernia. If a hernia can be reduced, then in no sense can there be an incarceration. I am now speaking of the power of taxis, such power as is possessed by the practiced surgical hand. And then I am led to say that a hernia is irreducible when with the skill of experience it can not be reduced. And then the condition of obstruction implies and involves the in-